Endovascular treatment of carotid stump syndrome

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Carotid stump syndrome is a rare but recognized cause of cerebrovascular events.[1] The syndrome is associated with carotid territory symptoms despite demonstration of occlusion of the ipsilateral internal carotid artery (ICA) on duplex ultrasound scans or angiograms. It is hypothesized that a residual stump of patent ICA is the source of emboli, which pass through the ipsilateral external carotid artery (ECA) into the middle cerebral artery circulation by way of reversed flow in the ophthalmic artery. To date all published cases of stump syndrome have been treated with surgical exploration and exclusion of the stump. We present the first case, to our knowledge, in which the stump was excluded with endovascular means with a covered stent.

CASE REPORT

A 61-year-old man underwent an uneventful left carotid endarterectomy at another vascular center in 1991. At presentation 11 years later he had a 4-week history of multiple (n = 17) left hemispheric transient ischemic attacks (TIA), with hemisensory and motor signs, usually in association with expressive dysphasia. Duplex scans showed that the ICA was occluded, but with a residual "stump" at the origin of the ICA. The contralateral ICA was 50% to 60% stenosed. In view of the fact that 3 weeks had elapsed since the most recent neurologic event, it was assumed that the ICA had recently become occluded. Risk factor management was optimized, clopidogrel therapy (75 mg/d) was started, and the patient was given an open appointment should any further problems occur.

The patient had no neurologic symptoms for 4 weeks before a second cluster of 9 left carotid territory TIA, with hemisensory or motor signs and dysphasia, within 14 hours. The patient denied any ocular symptoms. Systemic heparinization was instituted. Duplex scans confirmed the earlier findings, but in view of the recurrent symptoms, intra-arterial digital subtraction angiograms (IADSA) were obtained. Arch IADSA showed no evidence of inflow arterial disease in the arch or proximal common carotid artery. Selective IADSA (Fig 1) confirmed occlusion of the ICA and presence of a proximal ICA stump. It also demonstrated retrograde filling of the distal ICA through retrograde filling of the ophthalmic artery via branches of the ECA, confirming that the ECA was a collateral source of blood supply to the left hemisphere. There was no evidence of intracranial vascular disease and no evidence of filling of the left middle cerebral artery via transhemispheric crossover flow from the right carotid artery. Continuous transcranial Doppler ultrasound scanning was carried out for 45 minutes, and no emboli were detected in either middle cerebral artery. Functional imaging of the brain was not performed.

The differential diagnosis included embolization from the contralateral stenosed ICA via reversed flow in the left anterior cerebral artery,[2] embolization from the carotid stump to the ipsilateral intracranial circulation via the ECA branches and ophthalmic artery, hemodynamic TIA, and cardiac embolism. Embolization from the contralateral ICA stenosis was considered unlikely because not one cerebral event occurred ipsilateral to the right carotid stenosis. Hemodynamic TIA are usually associated with changes in posture, exercise, or light-headedness, none of which were noted in this patient. Similarly, cardiac embolism was considered unlikely because the patient had no signs, symptoms, or history of cardiac disease, and a chest x-ray film and transthoracic echocardiogram were normal. Most important, one would not have expected all 26 events to affect only one vascular territory in the brain. Accordingly, by process of exclusion, it was assumed that the most likely cause was embolization from the residual carotid stump. Treatment options thereafter included surgical or endovascular exclusion of the stump. In view of the patient's anatomy and because he had already undergone carotid surgery, endovascular exclusion was the preferred option.

The left common carotid artery was selectively catheterized with a 5F vertebral catheter (Terumo, Tokyo, Japan) via right common femoral artery puncture. Pre-reconstruction selective angiography confirmed a swirling contrast motion within the stump, which had an irregular superior surface (Fig 1). With roadmapping, an angled 0.035 hydrophilic guide wire (Terumo) was carefully manipulated into the ECA, averting any wire contact with the stump surface. An 8 × 50-mm wall graft endoprosthesis (Boston Scientific, Natick, Mass) was positioned across the stump and released. Measurements for sizing the prosthesis were taken from the diagnostic angiogram. A 6-mm × 4-cm angioplasty balloon (Smash; Boston Scientific) was dilated in the distal part of the wall graft to ensure adequate release. The wall graft (Fig 2) filled the tapered contours of the wider common carotid artery and narrower ECA, with no evidence of contrast material leakage into the excluded stump.

The patient was discharged home on the second postoperative day, but returned on the seventh postoperative day after having a brief episode of expressive dysphasia only, with no hemimotor or

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Competition of interest: none.
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J Vasc Surg 2003;38:593-5.

0741-5214/2003/$30.00 + 0
doi:10.1016/S0741-5214(03)00352-5

Fig 1. Intra-arterial digital subtraction angiogram of left carotid circulation. Internal carotid artery is occluded, but a residual stump *(black arrow)* is present. The distal internal carotid artery, however, fills via branches of the left external carotid artery through reversed flow in the ophthalmic artery *(white arrow)*.

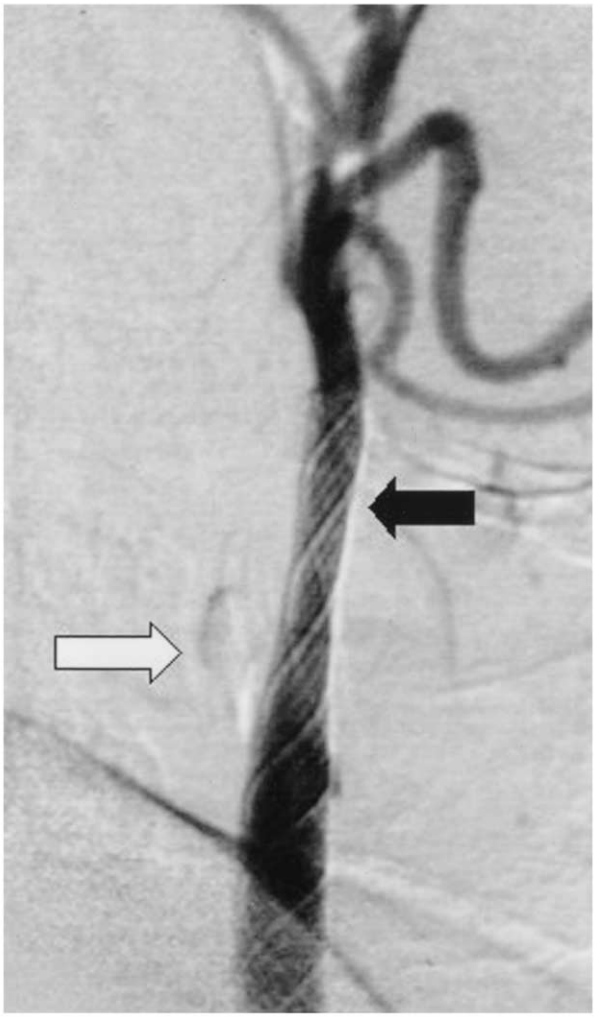

Fig 2. Intra-arterial digital subtraction angiogram of carotid bifurcation after endovascular exclusion of the stump. An 8 mm × 5 cm wall graft was positioned from the common carotid artery into the external carotid artery *(black arrow)*. The stent tapered naturally to fit comfortably, with no evidence of any contrast material leakage into the stump. The "ghost" of the excluded internal carotid artery stump is just visible *(white arrow)*.

sensory symptoms, which resolved within 30 minutes. Duplex ultrasound scans showed no abnormality within or adjacent to the wall graft and transcranial Doppler ultrasound scans again showed no evidence of ongoing embolization. In view of this, systemic anticoagulation was performed. Subsequent review at 1 and 3 months after stump exclusion found the patient to be entirely asymptomatic. Duplex ultrasound scans confirmed patency of the ECA, with no evidence of any contrast leakage around the prosthesis or into the stump.

DISCUSSION

Stump syndrome is a rare cause of cerebral vascular events,[1] but should be considered in the occasional patient with recurrent symptoms in the presence of ipsilateral ICA occlusion. Although sporadic cases have demonstrated the benefit of surgical exclusion of the stump, few large studies have been reported,[3] and many vascular surgeons have probably never seen or treated the condition. In Leicester, more than 1200 carotid endarterectomies have been performed over the last decade, but only two cases of stump syndrome have been treated surgically.

The current case typifies many of the diagnostic and management problems confronting the surgeon. First, it was not possible to be absolutely sure, before intervention, whether the cause was stump syndrome, transhemispheric embolization via reversed flow in the left anterior cerebral artery, hemodynamic, or cardioembolic. Angiography did, however, exclude significant disease in the aortic arch and proximal common carotid artery. The right (contralateral) ICA was 50% to 60% stenosed, and although there was some evidence of crossover flow into the left anterior cerebral artery, IADSA did not demonstrate filling of the

left middle cerebral artery after selective injection of contrast medium into the right carotid artery. Cardiac embolism remained a possibility, but one would not have expected all of the cerebral events to be localized to one vascular territory. Accordingly, it was concluded that stump syndrome was the most likely cause.

Having determined the most likely diagnosis, what was the optimal mode of treatment? The patient had had a second cluster of symptoms despite 4 weeks of clopidogrel therapy. Although formal anticoagulation remained an option, in view of the repeated events within such a short period it was decided to undertake stump exclusion. Therapeutic options included surgical or endovascular exclusion. Factors mitigating against a surgical approach included the patient's anatomy, ie, short, wide neck, and history of carotid endarterectomy. Both were believed to increase the risks for cranial nerve injury and operative stroke. The alternative was endovascular exclusion. To our knowledge, this is the first case in which a carotid stump has been excluded with endovascular placement of a covered stent.

Postoperatively the patient had another TIA, which resolved rapidly. We have not been able to demonstrate an embolic source for this, and it may have arisen from transient platelet thrombus at the proximal or distal limits of the wall graft. In view of this, formal anticoagulation was carried out. No other symptoms have occurred during follow-up, and the stent has remained patent with no evidence of any contrast material leakage into the stump or stenosis on duplex scan surveillance. Although efforts were made preoperatively to exclude an alternative cause to stump syndrome, that the patient had a further TIA after stent placement raises the question as to whether the diagnosis was correct and whether his symptoms might have stopped with anticoagulation therapy alone. Clearly, neither question can now be answered retrospectively. No further events have occurred, but this might also have been the situation if systemic warfarin therapy had not been introduced after stent insertion.

In conclusion, stump syndrome is a rare condition that has previously required treatment with surgical exploration and stump exclusion. Endovascular exclusion with a covered stent is, however, a new alternative to surgical intervention.

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Submitted Jan 28, 2003; accepted Feb 19, 2003.